Amendments to the Specification

Please replace the paragraph at page 1, lines 1 through 2 with the following amended paragraph:

METHODS OF TREATING ~~HEPATITIS~~ VIRAL INFECTION WITH CHIMERIC ANTI-TNF ANTIBODIES

Please replace the paragraph at page 25, lines 16-23 with the following amended paragraph:

As examples of antibodies according to the present invention, murine mAb A2 (ATCC Accession No. PTA-7045) of the present invention is produced by a cell line designated c134A. Chimeric antibody cA2 is produced by a cell line designated c168A. c134A was deposited pursuant to the Budapest Treaty requirements with the American Type Culture Collection (ATCC), 10801 University Boulevard, Manassas, Virginia 20110-220, on September 22, 2005. Cell line c134A is deposited as a research cell bank in the Centocor Cell Biology Services Depository, and cell line c168A(RCB) is deposited as a research cell bank in the Centocor Corporate Cell Culture Research and Development Depository, both at Centocor, 200 Great Valley Parkway, Malvern, Pennsylvania, 19355. The c168A cell line is also deposited at Centocor BV, Leiden, The Netherlands.

Please replace the paragraph at page 58, line 1 through page 59, line 14 with the following amended paragraph:

TNF related pathologies include, but are not limited to, the following:

(A) acute and chronic immune and autoimmune pathologies, such as systemic lupus erythematosus (SLE), rheumatoid arthritis, thyroidosis, graft versus host disease, scleroderma, diabetes mellitus, Graves' disease, Beschet's disease, and the like;

(B) infections, including, but not limited to, sepsis syndrome, cachexia, circulatory collapse and shock resulting from acute or chronic bacterial infection, acute and chronic parasitic and/or infectious diseases, bacterial, viral or fungal, such as a HIV, AIDS (including symptoms of cachexia, autoimmune disorders, AIDS dementia complex and infections);

(C) inflammatory diseases, such as chronic inflammatory pathologies and vascular inflammatory pathologies, including chronic inflammatory pathologies such as sarcoidosis, chronic inflammatory bowel disease, ulcerative colitis, and Crohn's pathology and vascular inflammatory pathologies, such as, but not limited to, disseminated intravascular coagulation, atherosclerosis, and Kawasaki's pathology:

(D) neurodegenerative diseases, including, but are not limited to, demyelinating diseases, such as multiple sclerosis and acute transverse myelitis; extrapyramidal and cerebellar disorders such as lesions of the corticospinal system; disorders of the basal ganglia or cerebellar disorders; hyperkinetic movement disorders such as Huntington's Chorea and senile chorea; drug-induced movement disorders, such as those induced by drugs which block CNS dopamine receptors; hypokinetic movement disorders, such as Parkinson's disease; Progressive supranucleo palsy; Cerebellar and Spinocerebellar Disorders, such as astructural lesions of the cerebellum; spinocerebellar degenerations (spinal ataxia, Friedreich's ataxia, cerebellar cortical degenerations, multiple systems degenerations (Mencel, Dejerine-Thomas, Shi-Drager, and MachadoJoseph)); and systemic disorders (Refsum's disease, abetalipoprotemia, ataxia, telangiectasia, and mitochondrial multi-system disorder); demyelinating core disorders, such as multiple sclerosis, acute transverse myelitis; disorders of the motor unit, such as neurogenic muscular atrophies (anterior horn cell degeneration, such as amyotrophic lateral sclerosis, infantile spinal muscular atrophy and juvenile spinal muscular atrophy);Alzheimer's disease; Down's Syndrome in middle age; Diffuse Lewy body disease; Senile Dementia of Lewy body type; Wernicke-Korsakoff syndrome; chronic alcoholism; Creutzfeldt-Jakob disease; Subacute sclerosing panencephalitis, Hallerrorden-Spatz disease; and Dementia pugilistica, or any subset thereof;

(E) malignant pathologies involving TNF-secreting tumors or other malignancies involving TNF, such as, but not limited to leukemias (acute, chronic myelocytic, chronic lymphocytic and/or myelodyspastic syndrome); lymphomas (Hodgkin's and non-Hodgkin's lymphomas, such as malignant lymphomas (Burkitt's lymphoma or Mycosis fungoides)); carcinomas (such as colon carcinoma) and metastases thereof; cancer-related angiogenesis; infantile haemangiomas;

(F) ~~hepatitis, e.g.,~~ alcohol-induced hepatitis; and

(G) other diseases related to angiogenesis or VEGF/VPF, such as ocular neovascularization, psoriasis, duodenal ulcers, angiogenesis of the female reproductive tract.

Please replace the paragraph at page 86, line 26 to page 87, line 12 with the following amended paragraph:

The complete primary sequence of human TNFα, according to Pennica *et al.*, *Nature 312*:724-729 (1984) is shown in Figure 13 (SEQ ID NO:1). Overlapping decapeptides beginning with every second amino acid and covering the entire amino acid sequence of human TNF-α were synthesized on polyethylene pins using the method of ~~Gysen~~ Geysen (~~Gysen~~ Geysen *et al.*, *Peptides: Chemistry and Biological*, Proceedings of the Twelfth American Peptide Symposium, p. 519-523, Ed, G.R. Marshall, Escom, Leiden, 1988). Sets of peptide pins bearing free N-terminal amino groups and acetylated N-terminal amino groups were individually prepared. Both sets of peptide pins were incubated in solutions containing the anti-TNF mAb cA2 to determine the amino acid sequences that make up the cA2 epitope on human TNF-α, as described below. Figure 14A shows the results of binding to the overlapping decapeptides that comprise the entire sequence of human TNFα. The O.D. (optional density) correlates directly with the increased degree of cA2 binding. Figure 14B shows the results of binding of cA2 to the same set of peptide pins in the presence of human TNFα. This competitive binding study delineates peptides which can show non-specific binding to CA2.